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FILE 'BIOSIS' ENTERED AT 14:07:45 ON 19 AUG 1997

L1 363 S HIV AND PLASMID
L2 19 S SIV AND PLASMID
L3 43 S SIV AND VECTOR
L4 40 S L3 NOT L2

=> t 13 bib ab 43 42 35 26

L3 ANSWER 43 OF 43 BIOSIS COPYRIGHT 1997 BIOSIS
AN 89:479169 BIOSIS
DN BA88:114929
TI THE GAG PRECURSOR OF SIMIAN IMMUNODEFICIENCY VIRUS ASSEMBLES INTO VIRUS-LIKE PARTICLES.
AU DELCHAMBRE M; GHEYSEN D; THINES D; THIRIART C; JACOBS E; VERDIN E; HORTH M; BURNY A; BEX F
CS DEP. MOL. BIOL., UNIV. BRUSSELS, BELGIUM.
SO EMBO (EUR MOL BIOL ORGAN) J 8 (9). 1989. 2653-2660. CODEN: EMJODG
ISSN: 0261-4189

LA English
AB To examine the potential role of the GAG precursor polyprotein in morphogenesis and assembly of the simian immunodeficiency virus (***SIV***), we have expressed the gag gene of SIVMac using a baculovirus expression ***vector*** . Infection of insect cells with recombinant virus containing the entire gag gene results in high expression of the GAG precursor protein, Pr57gag. The recombinant protein is myristylated and is released in the culture supernatant in an insoluble particulate form. A point mutation in the N-terminal glycine codon (Gly .fwdarw. Ala) inhibits myristylation. This mutated product is highly expressed but is not found in the culture supernatant. Electron microscopy and immunogold labelling of infected cells show that the native Pr57gag protein assembles into 100-120 nm virus-like particles that bud from the cell plasma membrane and are released in the culture supernatant. The unmyristylated protein also assembles into particulate structures which only accumulate inside the cells. These results demonstrate that the unprocessed GAG precursor of ***SIV*** can spontaneously assemble into particles in the absence of other viral proteins. Myristylation of the Pr57gag precursor is necessary for its association with the cell plasma membrane, for budding and for extracellular release.

L3 ANSWER 42 OF 43 BIOSIS COPYRIGHT 1997 BIOSIS
AN 90:414025 BIOSIS
DN BA90:74826
TI COEXPRESSION OF BIOLOGICALLY ACTIVE SIMIAN IMMUNODEFICIENCY VIRUS ***SIV*** REV AND ENV IN AN SV-40 SYSTEM THE ***SIV*** REV GENE REGULATES ENV EXPRESSION.
AU CHENG S-M; BLUME M; LEE S-G; HUNG P P; HIRSCH V M; JOHNSON P R
CS BIOTECHNOL. MICROBIOL. DIV., WYETH-AYERST RES., RADNOR, PENNSYLVANIA 19087.
SO VIROLOGY 177 (2). 1990. 816-819. CODEN: VIRLAX ISSN: 0042-6822
LA English
AB The coexpression of biologically active simian immunodeficiency virus (***SIV***) Rev and Env gene products was obtained in COS-1 cells from a single ***SIV*** subgenomic segment (which contains both

exons of rev and the entire env gene) cloned into a SV40-directed ***vector*** . The SIVsm Rev trans-activated the expression of the full-length env mRNA and was required for the production of envelope glycoproteins. Furthermore, the alignment of the structural conservation of the Rev functional domains among all HIV and ***SIV*** was analyzed.

L3 ANSWER 35 OF 43 BIOSIS COPYRIGHT 1997 BIOSIS
AN 93:58010 BIOSIS
DN BA95:34312
TI COEXPRESSION OF THE SIMIAN IMMUNODEFICIENCY VIRUS ENV AND REV PROTEINS BY A RECOMBINANT HUMAN ADENOVIRUS HOST RANGE MUTANT.
AU CHENG S-M; LEE S-G; RONCHETTI-BLUME M; VIRK K P; MIZUTANI S; EICHBERG J W; DAVIS A; HUNG P P; HIRSCH V M; ET AL
CS BIOTECHNOLOGY MICROBIOLOGY DIVISION, WYETH-AYERST RESEARCH, P.O. BOX 8299, PHILADELPHIA, PA. 19101.
SO J VIROL 66 (11). 1992. 6721-6727. CODEN: JOVIAM ISSN: 0022-538X
LA English
AB Recombinant human adenoviruses (Ads) that replicate in the intestinal tract offer a novel, yet practical, means of immunoprophylaxis against a wide variety of viral and bacterial pathogens. For some infectious agent such as human immunodeficiency virus (HIV), the potential for residual infectious material in vaccine preparations must be eliminated. Therefore, recombinant human Ads that express noninfectious HIV or other microbial proteins are attractive vaccine candidates. To test such an approach for HIV, we chose an experimental model of AIDS based on simian immunodeficiency virus (***SIV***) infection of macaques. Our data demonstrate that the ***SIV*** Env gene products are expressed in cultured cells after infection with a recombinant Ad containing both ***SIV*** env and rev genes. An E3 deletion ***vector*** derived from a mutant of human Ad serotype 5 that efficiently replicates in both human and monkey cells was used to bypass the usual host range restriction of Ad infection. In addition, we show that the ***SIV*** rev gene is properly spliced from a single ***SIV*** subgenomic DNA fragment and that the Rev protein is expressed in recombinant Ad- ***SIV*** -infected human as well as monkey cells. The expression of ***SIV*** gene products in suitable live Ad vectors provide an excellent system for studying the regulation of ***SIV*** gene expression in cultured cells and evaluating the immunogenicity and protective efficacy of ***SIV*** proteins in macaques.

L3 ANSWER 26 OF 43 BIOSIS COPYRIGHT 1997 BIOSIS
AN 94:219896 BIOSIS
DN 97232896
TI Expression of biologically active envelope glycoprotein from the acutely pathogenic simian immunodeficiency virus ***SIV*** -smmPBj.
AU Gonzalez S A; Affranchino J L; Burny A
CS Dep. Mol. Biol., Univ. Brussels, ULB, 1640 Rhode-St.-Genese, BEL
SO Virus Genes 8 (1). 1994. 75-78. ISSN: 0920-8569
LA English
AB The full-length envelope (env) gene from the most acutely pathogenic primate lentivirus described so far, the simian immunodeficiency virus ***SIV*** -smmPBj14 was expressed by a recombinant vaccinia virus ***vector*** (vv-env4) and was completely characterized as a previous step for its use as an immunogen in vaccination trials. Radioimmunoprecipitation and Western blot experiments indicated that

SIV -smmPBj gp160 precursor was processed into gp120 and gp41 subunits, and that gp120 was released into the medium. Flow cytometry analysis showed that recombinant ***SIV*** -smmPBj was transported to and expressed on the surface of venv4-infected cells. Biochemical analysis of virus-like particles produced by coinfection of cells with recombinant vaccinia viruses expressing ***SIV*** -smmPBj Env (vv-env4) and Gag (vv-wtgag) proteins revealed that the Env glycoprotein was incorporated into core-like particles. Furthermore, cells expressing ***SIV*** -smmPBj env gene products were found to undergo fusion with the same CD4+ cell lines in which the whole provirus has been shown to form syncytia.

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RECORDS LAST ADDED: 11 August 1997 (970811/ED)
CAS REGISTRY NUMBERS (R) LAST ADDED: 11 August 1997 (970811/UP)

=> s hiv and plasmid

66405 HIV
49048 PLASMID
L1 363 HIV AND PLASMID

8/19/97

=> t 11 bib ab 251 357 328 319 286 262 253 264 260

L1 ANSWER 251 OF 363 BIOSIS COPYRIGHT 1997 BIOSIS
AN 92:4461 BIOSIS
DN BA93:4461
TI FUNCTIONAL ANALYSIS OF LONG TERMINAL REPEATS DERIVED FROM FOUR STRAINS OF SIMIAN IMMUNODEFICIENCY VIRUS SIV-A-G-M IN RELATION TO OTHER PRIMATE LENTIVIRUSES.
AU SAKURAGI J-I; FUKASAWA M; SHIBATA R; SAKAI H; KAWAMURA M; AKARI H; KIYOMASU T; ISHIMOTO A; HAYAM M; ADACHI A
CS DEP. VIRAL ONCOL., INST. VIRUS RES., KYOTO UNIV., KYOTO 606, JPN.
SO VIROLOGY 185 (1). 1991. 455-459. CODEN: VURLAX ISSN: 0042-6822
LA English
AB The promoter activity of long terminal repeats (LTRs) of four strains of the simian immunodeficiency virus isolated from African green monkeys (SIVAGM) was compared with those of various LTRs derived from the other representative primate lentiviruses: human immunodeficiency virus type 1 (***HIV*** -1), type 2 (***HIV*** -2), SIV from a rhesus monkey (SIVMAC), and SIV from a mandrill (SIVMND). The expression of the LTRs was evaluated by monitoring chloramphenicol acetyltransferase production after transfection of reporter ***plasmid*** clones. In the absence of viral tat, all SIVAGM LTRs acted as much more efficient promoters than any of the other LTRs. When tat gene products were supplied in trans, LTRs of SIVAGM and SIVMND were activated inefficiently relative to high responder LTRs of ***HIV*** -2 and SIVMAC. The LTR of ***HIV*** -1 was highly activated by ***HIV*** -1 tat, but not so much by ***HIV*** -2, SIVAGM, and SIVMND tat.

L1 ANSWER 357 OF 363 BIOSIS COPYRIGHT 1997 BIOSIS
AN 88:177493 BIOSIS
DN BA85:89595
TI COEXPRESSION OF HUMAN IMMUNODEFICIENCY VIRUS ENVELOPE PROTEINS AND TAT FROM A SINGLE SV-40 LATE REPLACEMENT VECTOR.
AU REKOSH D; NYGREN A; FLODBY P; HAMMARSKJOLD M-L; WIGZELL H
CS DEP. BIOCHEM., STATE UNIV. N.Y. BUFFALO, BUFFALO, N.Y. 14214.
SO PROC NATL ACAD SCI U S A 85 (2). 1988. 334-338. CODEN: PNASA6 ISSN: 0027-8424
LA English
AB A Sal I-Xba I fragment containing the genes encoding tat, art, and the envelope proteins from the BH10 clone of human immunodeficiency virus (***HIV***) was inserted into a simian virus 40 (SV40)-based eukaryotic expression vector. The vector is a shuttle vector that replicates to high copy numbers in both Escherichia coli and eukaryotic cells permissive for SV40 replication. Transfection of the ***HIV*** DNA-containing vector (pSVSX1) into the CV-1 monkey cell line gave high levels of expression of the envelope glycoproteins gp160 and gp120 in 20-30% of the transfected cells. By several criteria, the proteins were indistinguishable from those produced during infection. The proteins were localized to the cytoplasm and plasma membrane, and some of the gp120 was shed into the culture medium. Approximately 0.5 .mu.g of envelope protein could be extracted from 106 cells. This is at least 100 times higher than the levels found in ***HIV*** -infected H9 cells. In addition, a trans-activation assay performed with pSVSX1 and a ***plasmid*** containing the gene for chloramphenicol acetyltransferase under the

control of the ***HIV*** long terminal repeat demonstrated that a functional tat gene product also was expressed. Thus, this transient vector system provides an abundant source of native envelope protein for purification and characterization and also will be useful for studies dealing with the regulation of ***HIV*** gene expression.

L1 ANSWER 328 OF 363 BIOSIS COPYRIGHT 1997 BIOSIS
AN 89:403927 BIOSIS
DN BA88:73352
TI HUMAN CELL LINES STABLY EXPRESSING ***HIV*** ENV AND TAT GENE PRODUCTS.
AU GAMA SOSA M A; DEGASPERI R; FAZELY F; RUPRECHT R M
CS DEP. PATHOL., HARVARD MED. SCH., BOSTON, MASS. 02115.
SO BIOCHEM BIOPHYS RES COMMUN 161 (1). 1989. 305-311. CODEN: BBRCA9
ISSN: 0006-291X
LA English
AB A DNA fragment containing the tat, rev and env genes of the human immunodeficiency virus type 1 was inserted into the retroviral vector pZIPneoAU3. The resulting ***plasmid*** penvAU3 was transfected into HeLa and .psi.CRIP cells. Resulting recombinant retroviruses were used to infect HeLa and Jurkat cells. Immunoprecipitation analysis of stable transformants showed the expression of ***HIV*** env glycoproteins gp160, gp120 and gp41. Transactivation assays with a ***plasmid*** containing the gene for chloroamphenicol acetyltransferase linked to ***HIV*** promoter-enhancer sequences demonstrated the expression of functional tat. These cells constitute virus-free tools for functional and structural studies of native env and tat.

L1 ANSWER 319 OF 363 BIOSIS COPYRIGHT 1997 BIOSIS
AN 90:47577 BIOSIS
DN BA89:24941
TI ***PLASMID*** LIBRARY OR THE TRANSCRIPTION OF RNA PROBES COMPLEMENTARY TO THE ENTIRE GENOME OF THE HUMAN IMMUNODEFICIENCY VIRUS TYPE 1 ***HIV*** -1.
AU ROY C; KNAAK C; GARVIE P; LIMMER B; CAMPIONE-PICCARDO J
CS MOL. VIROL. LAB., LAB. CENT. DIS. CONTROL, HEALTH WELFARE CANADA, OTTAWA, ONT., CANADA K1A 0L2.
SO BIOCHEM CELL BIOL 67 (9). 1989. 510-515. CODEN: BCBIEQ ISSN: 0829-8211
LA English
AB A ***plasmid*** (pBH10R3) containing a 9-kb Sst I fragment of ***HIV*** -1 (clone BH-10) inserted in pSP64, an in vitro expression vector, has been used for the transcription of anti-sense ***HIV*** -1 RNA. With this system, the transcripts obtained in vitro were not usually full length (1 to 2 kb long) and they predominantly span the 3' end ORF and ENV regions of the viral genome. We have rearranged the ***HIV*** -1 genomic sequences with respect to the SP6 promoter in the pSP64 vector and have obtained a series of new constructs allowing the expression in vitro of RNA transcripts complementary to other regions in the ***HIV*** -1 genome, including the 5' end of the ENV region as well as the TAT, POL, and GAG regions. In fact, the combined use of these constructs as templates for in vitro transcription allows the production of RNA probes spanning the entire viral genome. Compared with the 1- to 2-kb probes mentioned above, the combined use of such probes results in a several-fold increase in the sensitivity of molecular hybridization for the detection of ***HIV*** -1 nucleic acid sequences. Also,

these constructs enable the preparation of RNA probes that have the potential to detect restriction polymorphisms throughout the ***HIV*** -1 genome.

L1 ANSWER 286 OF 363 BIOSIS COPYRIGHT 1997 BIOSIS
AN 91:33655 BIOSIS
DN BA91:23006
TI PRODUCTION AND CHARACTERIZATION OF A FRAGMENT CONTAINING THE ***HIV*** -GP120 BINDING REGION OF CD4 USING A BOVINE PAPILLOMA VIRUS BPV VECTOR.
AU GIDLUND M; SYDOW M; STENBECK A; FLODBY P; FOSSUM H; MATSUDA S; LUNDIN K; WIGZELL H; LIND P
CS KABIGEN AB, STRANDBERGSGATAN 49, S-112 87 STOCKHOLM, SWEDEN.
SO ARCH VIROL 113 (3-4). 1990. 209-220. CODEN: ARVIDF ISSN: 0304-8608
LA English
AB We have used a bovine papilloma virus (BPV) based mammalian cell expression vector consisting of the complete BPV genome and a human cytomegalovirus transcription unit for the production of soluble CD4. Mouse C-127 cells were transfected with vector DNA together with a selectable G418 resistance ***plasmid***. Surviving clones were selected for high production using a solid phase ELISA based on the immobilization of supernatant-derived CD4 onto nitrocellulose paper and subsequent detection with anti-CD4 antibodies. The expressed protein was shown to bind ***HIV*** -gp120 and efficiently block ***HIV*** -1 infection in vitro. The possibility to use the above system for rapid production of defined glycoprotein fragments harboring defined functional regions, for the further elucidation of the functional role of CD4 in antigen presentation and cell to cell contact, and for possible intervention during ***HIV*** infection is discussed.

L1 ANSWER 262 OF 363 BIOSIS COPYRIGHT 1997 BIOSIS
AN 91:454479 BIOSIS
DN BA92:99259
TI EXPRESSION OF THE ENV GENE FRAGMENT OF ***HIV*** -1 IN ESCHERICHIA-COLI CELLS.
AU KAZENNOVA E V; BOBKHOVA M R; BOBKOV A F; SERGEEV O V; LUKASHEVICH N V; GARAEV M M
CS D.I. IVANOVSKII INST. VIROL., ACAD. MED. SCI. USSR, MOSCOW, USSR.
SO VOPR VIRUSOL 36 (2). 1991. 122-125. CODEN: VVIRAT ISSN: 0507-4088
LA Russian
AB A recombinant ***plasmid*** pEK6 determining the synthesis of a hybrid protein the N-terminus of which was represented by full-size beta-galactosidase and C-terminus by ***HIV*** -1 gene env virus-specific sequence was constructed. The analysis of lysates of *E. coli* HB101/pEK6 bacteria in 6% PAAGE revealed additional proteins with molecular weights from 185 to 130 kDa. These proteins interacted with blood serum antibodies of a virus carrier but formed no specific bands with sera from normal donors. Densitometric analysis of polyacrylamide gels stained with Coomassi R250 demonstrated that the level of production of recombinant protein was at least 15% of the total cell protein. Hybrid polypeptides formed poorly soluble inclusion bodies in the bacterial cells. Study of the immunological properties of the recombinant polypeptides showed that immunization of rabbits with these proteins induced antibodies specifically reacting with viral polypeptides with molecular weights of about 82 and 140 kDa. Such features as a high level of synthesis, technologically feasible purification of inclusion bodies, and

adequate antigenic properties recommend this preparation for use in the development of diagnostic test systems.

L1 ANSWER 253 OF 363 BIOSIS COPYRIGHT 1997 BIOSIS
AN 91:531377 BIOSIS
DN BA92:142837
TI SOLUBLE CD4-PE40 IS CYTOTOXIC FOR A TRANSFECTED MAMMALIAN CELL LINE STABLY EXPRESSING THE ENVELOPE PROTEIN OF HUMAN IMMUNODEFICIENCY VIRUS ***HIV*** -1 AND CYTOTOXICITY IS VARIABLY INHIBITED BY THE SERA OF ***HIV*** -1 INFECTED PATIENTS.
AU PITTS T W; BOHANON M J; LEACH M F; MCQUADE T J; MARSCHKE C K; MERRITT J A; WIERENGA W; NICHOLAS J A
CS DEP. CANCER INFECTIOUS DISEASES, UPJOHN LAB., KALAMAZOO, MICH. 49007.
SO AIDS RES HUM RETROVIRUSES 7 (9). 1991. 741-750. CODEN: ARHRE7 ISSN: 0889-2229
LA English
AB Sera were obtained from 50 individuals infected with human immunodeficiency virus type 1 or from ***HIV*** -1-uninfected individuals before or after vaccination with recombinant gp 160. These sera were evaluated for activity antagonistic to the cell-killing activity of the chimeric *Pseudomonas* exotoxin hybrid protein, sCD4-PE40. For these studies, Chinese hamster ovary (CHO) cells were transfected with a chimeric ***plasmid*** encoding the tat, rev, and envelope genes of ***HIV*** -1 and a cell line was selected for stable expression of the envelope glycoproteins at the cell surface (CHO-env). Cytotoxicity of sCD4-PE40 for CHO-env in the presence or absence of added human serum was quantitated spectrophotometrically following enzymatic reduction of a tetrazolium bromide within the mitochondria of viable cells (MTT assay). Several ***HIV*** + sera inhibited the cytotoxic activity of sCD4-PE40; the antagonist had properties consistent with those of immunoglobulins in that it was heat stable, absorbed by protein A, and reversible by increasing the concentration of sCD4-PE40. Of 15 ***HIV*** + sera which strongly reacted with gp120, 11 (73%) also potently inhibited sCD4-PE40 cytotoxicity, and cytotoxicity was inhibited by sera from some ***HIV*** - individuals after, but not before, immunization with gp160. These data suggested a role for antibody to gp120 in the antagonistic activity. However, not all sera with antibody to gp120 antagonized sCD4-PE40 cytotoxicity and high levels of antagonist activity were frequently (40%) found in ***HIV*** + sera lacking immunoblot-detectable antibody to gp120, or antibody to either CD4 or PE40. Grouping of the ***HIV*** + sera according to the patients' absolute number of CD4+ cells revealed that the degree of inhibition of sCD4-PE40 cytotoxicity approached a Gaussian distribution, suggesting that persons with CD4+ cell counts between 200 and 700/mm³ may be more likely to possess significant levels of serum antagonist. These data have implications for the clinical development of sCD4-PE40 or other sCD4-based therapeutics in the management of ***HIV*** -1 infection.

L1 ANSWER 264 OF 363 BIOSIS COPYRIGHT 1997 BIOSIS
AN 91:424509 BIOSIS
DN BR41:74054
TI A NEW ***PLASMID*** CONSTRUCT AS INTERNAL STANDARD FOR ***HIV*** PCR.
AU TELENTI A; IMBODEN P
CS INST. MED. MICROBIOLOGY, UNIV. BERNE, SWITZ.
SO ISTITUTO SUPERIORE DI SANITA. VII INTERNATIONAL CONFERENCE ON AIDS:

SCIENCE CHALLENGING AIDS; FLORENCE, ITALY, JUNE 16-21, 1991.
464P. (VOL. 1); 460P. (VOL. 2). ISTITUTO SUPERIORE DI SANITA: ROME,
ITALY. PAPER. 0 (0). 1991. 118.

DT Conference
LA English

L1 ANSWER 260 OF 363 BIOSIS COPYRIGHT 1997 BIOSIS
AN 91:455532 BIOSIS
DN BA92:100312
TI INTERACTION OF CD4 WITH HLA CLASS II ANTIGENS AND ***HIV***
GP120.
AU PIATIER-TONNEAU D; GASTINEL L-N; AMBLARD F; WOJCIK M; VAIGOT P;
AUFFRAY C
CS INST. EMBRYOL. CELLULAIRE MOL. CNRS, 49 BIS, AVENUE DE LA BELLE
GABRIELLE, F-94130 NOGENT-SUR-MARNE, FR.
SO IMMUNOGENETICS 34 (2). 1991. 121-128. CODEN: IMNGBK ISSN: 0093-7711
LA English
AB We have developed a cellualr adhesion assay in which B lymphocytes
expressing HLA class II antigens form rosettes with COS cells
expressing high levels of cell surface CD4 upon transient
transfection with a CDM8-CD4 ***plasmid*** construct. The assay
is specific, quantitative, and overcomes the difficulties encountered
with a previously described system using an SV40 viral vector.
Rosette formation was inhibited by a series of CD4-and
HLA-DR-specific antibodies, as well as by human immunodeficiency
virus (***HIV***) gp 120, and a synthetic peptide derived from
part of its binding site for CD4 (amino acid residues 414-434), but
not by a variety of other effectors, inlcuding several soluble CD4
derivatives. The comparison of this pattern of inhibition with those
observed in other systems further emphasizes the great similarity,
but incomplete identity, in the CD4 binding sites for HLA class II
antigens and ***HIV*** gp120, and supports a model in which CD4
is considered as an allosteric servo modulator of T-cell adhesion and
function which probably is induced to interact with HLA class II
antigens when associated with the Tcr/CD3 complex.

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